

Improved Regional Ventricular Function After Successful Surgical Revascularization

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Left ventricular segments with reversible asynergy at rest demonstrate reversible myocardial perfusion defects on exercise thallium-201 scintigrams. To determine if improved perfusion eliminates asynergy at rest, 23 patients with angina (stable in 21, unstable in 2) were studied before and after coronary artery bypass surgery. All patients underwent exercise myocardial perfusion scintigraphy, contrast ventriculography and coronary arteriography before and after surgery. Selective graft angiography was performed during the postoperative catheterization to determine graft patency. Segmental ventricular function was quantitated by a regional fraction method. The scintigrams were divided into five regions and compared with the corresponding regions of the ventriculogram.

Seventy-one of a possible 142 ventricular segments exhibited exercise-induced perfusion deficits. Preoperative regional ejection fraction was normal in 42 of these

segments and abnormal in 29. Postoperatively, in 19 of the abnormal segments, function improved or normalized. All these segments had improved perfusion during exercise after surgery and were supplied by a patent bypass graft. Nine of the 10 segments in which abnormal wall motion persisted postoperatively continued to have exercise-induced perfusion deficits, and 9 of the 10 segments were supplied by an occluded or stenotic graft or one with poor run off. Of the 42 segments with normal wall motion preoperatively, 30 had improved perfusion after surgery and 35 maintained normal function.

This study indicates that asynergy at rest is permanently reversed after coronary bypass surgery if improved myocardial perfusion can be documented. These findings are consistent with but do not prove the concept that reversible rest asynergy may reflect chronic ischemia or a prolonged effect from previous ischemic episodes.

Infarction of myocardium with the subsequent development of fibrosis produces irreversible ventricular wall motion abnormalities (1) and myocardial scintigraphy with thallium-201 usually demonstrates a persistent perfusion defect in the same region (2). However, patients without clinical evidence of myocardial infarction, including some with stable angina pectoris, may have abnormal wall motion in the resting state (3). Although rest asynergy may temporarily improve after the administration of nitroglycerin or epi-

nephine or after a premature ventricular contraction, its cause is unknown (4-6).

Previous work from our laboratory (7) has shown that left ventricular segments with temporarily reversible wall motion abnormalities often have exercise-induced thallium-201 perfusion deficits in the same regions. These findings suggested to us that some wall motion abnormalities at rest may result from persistent silent ischemia or prolonged functional injury from previous ischemic episodes. We hypothesized that treatment that produced better perfusion might result in permanent improvement in wall motion. However, previously published studies (8-11) examining the effect of coronary artery bypass surgery on segmental wall motion, particularly in patients with stable angina, have yielded conflicting results. Furthermore, there is no study of patients with stable angina that has examined perfusion, quantitated changes in wall motion and determined graft patency. Therefore, we studied myocardial perfusion by thallium-201 scintigraphy and segmental wall motion by contrast ventriculography both before and several months after bypass surgery.

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In addition, graft status was determined by angiography to ascertain if successful revascularization with documented improvement in perfusion resulted in better contraction.

Methods

Patient selection. The records of all patients who had technically satisfactory exercise redistribution thallium-201 scintigraphy, contrast ventriculography and coronary angiography before and after coronary bypass surgery were reviewed. Patients without a reversible perfusion defect on the preoperative scintigram or less exercise effort during the postoperative stress test than during the preoperative test were excluded. All studies were completed between December 1975 and October 1980. During the study period, patients with reversible preoperative perfusion defects were invited to have elective postoperative cardiac catheterization and exercise myocardial scintigraphy, but in most patients in this study recatheterization was performed for clinical indications such as recurrence of chest pain.

Patient characteristics. The study group comprised 23 patients. Their mean age was 54.7 ± 6.7 years; 19 were male and 4 were female. The indication for surgery was angina unresponsive to medical treatment in all cases. The angina pattern was stable up to the time of surgery in 21 patients. Two patients developed an unstable angina syndrome (rest pain) in the short interval (<6 weeks) between their stress test and surgery. The patients received a total of 65 grafts for an average of 2.8 ± 0.7 grafts per patient; they underwent catheterization an average of 49.2 ± 77.1 days before surgery and a mean of 255.0 ± 151.9 days after surgery. Preoperatively, 15 patients were receiving beta-adrenergic blocking drugs and 16 were taking nitrates. Only one patient was not receiving drug therapy before surgery. In addition, two patients were taking digoxin, six were taking diuretic drugs, two were taking antiarrhythmic agents and three were taking antihypertensive medication. Postoperatively, six patients were taking no medication, six were taking beta-adrenergic blocking drugs and six were taking nitrates. In addition, 4 patients were taking digoxin, 10 were taking diuretic drugs and 5 were taking antihypertensive medication. Information on postoperative medication was incomplete in two patients.

Exercise myocardial perfusion scintigraphy. Exercise was performed before and after operation on a treadmill using a modified Bruce protocol (12). Blood pressure and 12 lead electrocardiograms were recorded at 90 second intervals during the test. Exercise was terminated with the development of limiting chest pain or when dyspnea, fatigue or other symptoms precluded continuing. Patients who, on post-surgical assessment, did not achieve their preoperative exercise duration or rate-pressure product were excluded from the study. All exercise stress tests were performed within 2 months of cardiac catheterization and the majority were

done within 1 week. Thallium-201, 1.5 to 2 mCi, was injected intravenously 1 minute before termination of the exercise. After completion of the postexercise monitoring period, myocardial scintigrams were performed with an Ohio Nuclear Series 120 or Searle Pho Gamma IV scintillation camera, using converging or linear collimation and a 20% window centered at approximately 75 keV. The initial image was taken in the anterior projection to 300,000 counts and subsequent left anterior oblique and left lateral projections were taken to equal time. Scintigraphy was completed within 30 minutes after injection. Patients with abnormal stress images were reimaged after 4 hours or restudied within 1 week at rest.

The unprocessed scintigrams were evaluated by two independent observers. The readers agreed initially in their assessment of 95% of the regions and were able to reach consensus in the remaining 5%. Exercise and rest scintigrams were first interpreted individually and then compared. A region was considered abnormal when decreased perfusion was present on the exercise scintigram. These abnormalities were compared with comparable regions on the rest scintigram and were further classified as unchanged, improved or normalized. Preoperative and postoperative studies were interpreted independently as well as blindly, and then compared.

Cardiac catheterization. Patients underwent catheterization in the postabsorptive state after sedation with 50 mg of meperidine and 25 mg of phenergan intramuscularly, or 10 mg of diazepam orally. Cineventriculograms were obtained by injecting 0.5 to 0.8 ml/kg of meglucamine diatrizoate through a pigtail catheter in 4 seconds and recording the images at 60 frames/second. All ventriculograms were done before coronary and graft angiography. Biplane ventriculograms in the 30° right anterior oblique and 60° left anterior oblique views were obtained in nine patients. Only the 30° right anterior oblique view was available in 14 patients.

Outlines of the left ventricular silhouette were traced at end-systole and end-diastole. End-diastole was defined as the largest ventricular outline before mitral valve closure, and end-systole as the smallest outline before the mitral opening. When biplane ventriculography was performed, the same beat was traced in the right anterior oblique and left anterior oblique views. Only sinus beats occurring within five contractions of contrast injections were used. Postectopic beats were excluded from analysis.

Graft patency was determined by selective angiography. Graft occlusion was confirmed by identifying a graft diverticulum with selective angiography or not demonstrating the graft by aortography.

Comparison of scintigrams and ventriculograms. Our method for quantitative segmental analysis of ventriculograms has been previously described in detail (13). We have also described a method for comparing similar anatomic

regions on contrast ventriculogram and myocardial scintigram (7). Succinctly, we divided the right anterior oblique ventriculographic outlines into five areas and the left anterior oblique silhouettes, when available, into three areas (Fig. 1). There were 142 segments available for regional wall motion analysis in this study, and an area ejection fraction was calculated for each segment. An area ejection fraction 2 standard deviations below the previously established mean of normal subjects (13) was considered abnormal.

The anterior scintigrams were divided into anterior, apical and inferior regions; the left lateral images were divided into anterior and inferior regions, and the left anterior oblique view into septal and posterolateral regions (Fig. 1). We compared scintigrams and ventriculograms in the following manner: anterior scintigraphic regions were related to ventriculographic areas 1 and 2; apical to area 3; inferior to areas 4 and 5; septal to area 8; and posterolateral to areas 5, 6, and 7.

A significant improvement in postoperative segmental wall motion was defined as an area ejection fraction increase from the abnormal to normal range of at least 1 standard deviation. An area ejection fraction increase of 2 standard deviations within the abnormal range was also considered to be a significant improvement.

Statistics. Paired data were analyzed by the one-tailed paired *t* test for significance. Probability (*p*) values less than 0.05 were considered significant. The correlation between wall motion and perfusion after surgery for all segments was evaluated by a one way analysis of variance. The re-

lation between postoperative wall motion and perfusion for the segments with abnormal wall motion before surgery was also evaluated by a chi-square analysis of trend. Data are expressed as mean values \pm standard deviation.

Results

Exercise myocardial perfusion scintigraphy. Before surgery, 71 of the 142 ventricular segments available for analysis exhibited a reversible perfusion defect (Table 1). The duration of treadmill exercise increased significantly ($p < 0.001$) from 5.7 ± 2.4 minutes before surgery to 7.6 ± 2.3 minutes after surgery. Similarly, the heart rate blood pressure product increased from $15.9 \times 10^3 \pm 4.5 \times 10^3$ to $19.2 \times 10^3 \pm 4.4 \times 10^3$ ($p < 0.005$).

Postoperative cardiac catheterization. The postoperative graft patency rate was 89% (58 of 65). A significant stenosis was noted in the body of three grafts and at the distal anastomosis in three others. Consequently, 52 (80%) of 65 grafts were considered to be functioning well at the time of restudy.

Every ventricular segment with abnormal wall motion preoperatively that exhibited improved (3 segments) or normal (15 segments) function postoperatively was perfused by a patent graft. In all but three instances, the grafts were found to be functioning well by selective graft angiography (Table 2).

Of the 10 asynergic ventricular segments that did not show improvement in regional function, 3 were associated with an occluded grafts, 2 with a stenosed graft and 4 with severe distal disease in the native coronary artery. In only one instance was lack of improvement in function associated with a good quality graft.

Global ejection fraction increased from 60.4 ± 14.5 preoperatively to 67.7 ± 11.0 after surgery ($p < 0.025$).

Comparison of scintigrams and ventriculograms. The 71 ventricular segments identified with reversible perfusion abnormalities before surgery were analyzed in detail (Table 1). Preoperative wall motion was normal in 42 segments and abnormal in 29. The great majority of segments with normal preoperative wall motion exhibited a normal (28 segments) or improved (1 segment) postoperative exercise perfusion pattern.

Approximately two-thirds (19 of 29) of the ventricular segments with abnormal preoperative wall motion showed improvement after surgery. In 16 of 19 instances, improvement in function was associated with normalization in scintigraphic perfusion (Fig. 2). Perfusion improved but was still abnormal in two segments and unchanged in one. All segments with improved perfusion were supplied by patent grafts.

In 10 segments, preoperative asynergy did not decrease. Postoperative perfusion, as assessed by scintigraphy, improved but failed to normalize in five segments and did not

Figure 1. For comparison, scintigrams were divided into five regions: anterior (ANT), apex, inferior (INF), septal (SEPT) and posterior (POST). Ventriculograms were divided into eight regions as numbered. LAO = left anterior oblique; RAO = right anterior oblique.

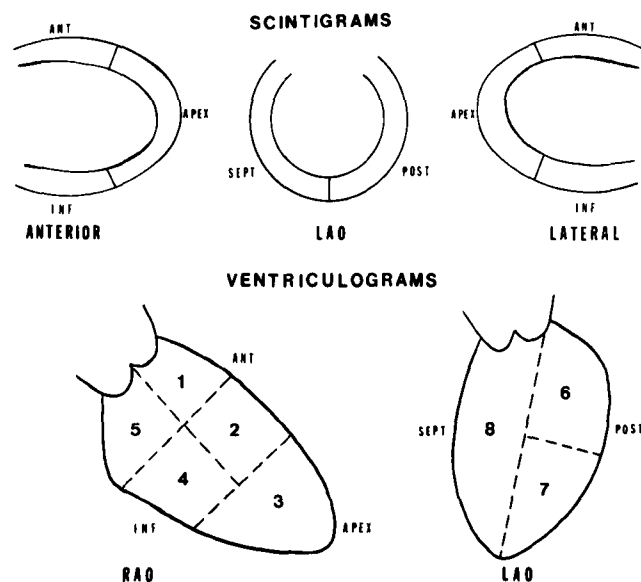


Table 1. Exercise-Induced Perfusion Defects in 71 Segments

| | | Normal Segmental Wall Motion | | | Abnormal Segmental Wall Motion | | |
|--------------------|--|------------------------------|---------------|---------------|--------------------------------|---------------|---------------|
| Preop wall motion | | 42 | | | 29 | | |
| n | | (0.50 ± 0.14) | | | (0.22 ± 0.10) | | |
| SEF | | | | | | | |
| Postop wall motion | | Normal | Decreased | Abnormal | Normal | Improved | Abnormal |
| n | | 35 | 3 | 4 | 16 | 3 | 10 |
| SEF | | (0.56 ± 0.14) | (0.46 ± 0.04) | (0.24 ± 0.05) | (0.48 ± 0.08) | (0.28 ± 0.02) | (0.24 ± 0.11) |
| Postop perfusion | | ↑ → ↓ | ↑ → ↓ | ↑ → ↓ | ↑ → ↓ | ↑ → ↓ | ↑ → ↓ |
| n | | 29 6 0 | 2 0 1 | 3 1 0 | 16 0 0 | 2 1 0 | 6 4 0 |
| SEF | | (0.58 ± 0.14) | (0.46 ± 0.05) | (0.22 ± 0.07) | (0.48 ± 0.08) | (0.29 ± 0.02) | (0.24 ± 0.07) |
| | | (0.47 ± 0.09) | (0.45) | (0.31) | (0.27) | (0.24 ± 0.15) | |

n = number of segments; Postop = postoperative; Preop = preoperative; SEF = segmental ejection fraction; ↑ = increased; → = unchanged; ↓ = decreased.

improve at all in four segments (Fig. 3). Two of the four segments that did not demonstrate improvement in perfusion were supplied by a stenosed graft and two were associated with severe disease in the native coronary artery distal to the graft insertion point (Table 2). In the remaining six segments, perfusion improved but did not normalize in five. Of these five segments, three were associated with graft occlusion and two were associated with severe disease in the native vessel. There was only one segment with unchanged abnormal wall motion after surgery in which perfusion normalized and the graft was patent (Table 2). A chi-square test for trend for the 29 segments demonstrating abnormal preoperative wall motion showed a positive correlation between improved wall motion and improved per-

fusion postoperative (χ^2 trend = 6.93, $p < 0.01$). A one way analysis of variance for all 71 segments also showed a strong correlation between improved regional rejection fraction and improved postoperative perfusion (F statistic = 13.96, $p < 0.01$).

Overall, there were only 12 segments in which postoperative function and scintigraphic perfusion appeared to be discordant (Table 1). In eight segments however, although perfusion improved it was still abnormal after surgery. When graft status was considered, only 3 of the 12 segments were adequately revascularized. Therefore, improvement in perfusion usually correlated with preservation of normal function or improvement in abnormal function. Failure to improve abnormal function usually correlated with little or no

Table 2. Graft Patency

| Postoperative Wall Motion | Postoperative Perfusion (no. of segments) | Graft Patency | Quality of Revascularization |
|------------------------------------|--|---------------|--|
| Normal wall motion maintained | Improved (29) | 29/29 | 1 stenotic graft |
| | Unchanged (6) | 6/6 | 2 stenotic grafts, severe distal disease native vessel in 2 |
| | Total (35) | | |
| Normal wall motion decreased | Improved (2) | 1/2 | 1 segment not grafted |
| | Decreased (1) | 0/1 | Segment not grafted |
| | Total (3) | | |
| Normal wall motion became abnormal | Increased (3) | 3/3 | |
| | Unchanged (1) | 1/1 | Severe distal disease native vessel |
| | Total (4) | | |
| Abnormal wall motion normalized | Improved (16) | 16/16 | 3 stenotic grafts |
| | Total (16) | | |
| Abnormal wall motion improved | Improved (2) | 2/2 | |
| | Unchanged (1) | 1/1 | Severe distal disease native vessel |
| | Total (3) | | |
| Abnormal wall motion unchanged | Improved (6) | 3/6 | Severe distal disease native vessel in 2 |
| | Unchanged (4) | 4/4 | 2 stenotic grafts, severe distal disease native vessel in 2 |
| | Total (10) | | |

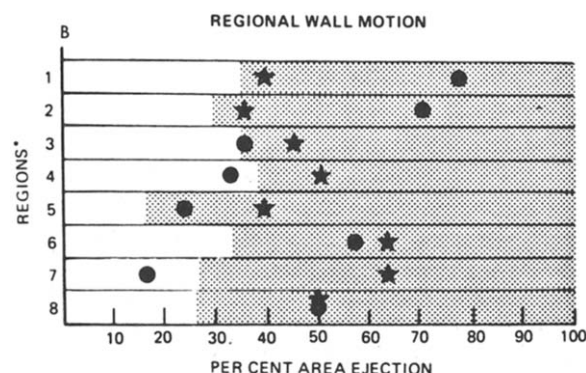
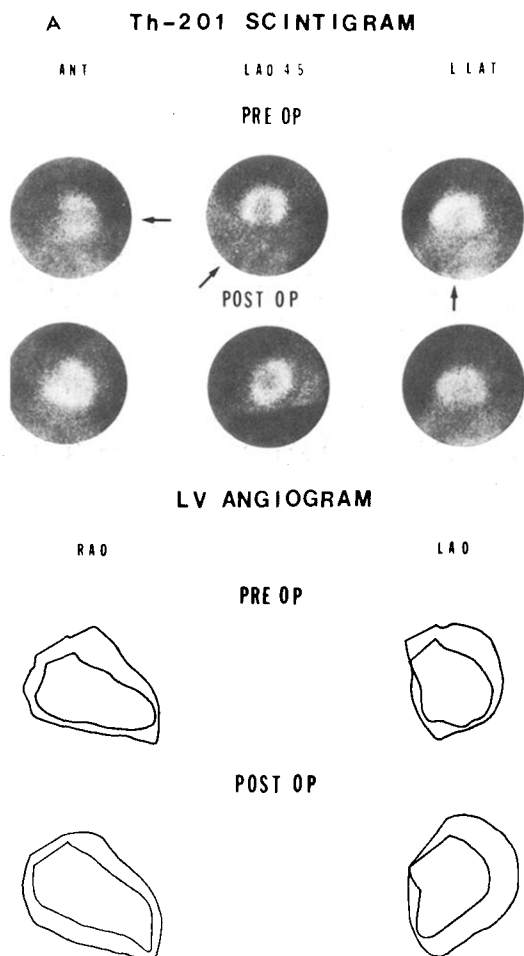


Figure 2. **A**, Comparison of preoperative (PRE OP) and postoperative (POST OP) exercise thallium-201 (Th-201) scintigrams and left ventriculographic silhouettes indicates improvement in inferior, posterior and apical perfusion (arrows) and inferior and posterior wall motion. Grafts to the left anterior descending and posterior descending coronary arteries were patent. ANT = anterior; LAO 45 = 45° left anterior oblique; L LAT = left lateral; RAO = right anterior oblique. **B**, Comparison of the results of preoperative (circles) and postoperative (stars) quantitative regional wall motion analysis of the ventriculographic silhouettes in **A** confirms objective improvement in regions 4 and 7. The **stippled area** represents the range of normal regional area ejection fraction.

improvement in perfusion and a poorly functioning or occluded graft.

Discussion

Our study provides convincing evidence that depressed but reversible segmental function at rest is permanently improved after surgical revascularization in patients with stable angina if the region is supplied by a well functioning graft with evidence of improved perfusion by thallium-201 scintigraphy. The majority of ventricular segments demonstrating abnormal wall motion and exercise-induced perfusion deficits before surgery had better or normal wall motion postoperatively when perfusion during exercise was entirely normal (16 [84%] of 19). All functionally improved segments were supplied by a patent graft. Asynergic segments that did not improve postoperatively continued to show some perfusion deficit in 9 of 10 instances, and only 1 was supplied by a patent unstenosed graft with a good native vessel. The relation between improved postoperative wall motion and perfusion was very significant ($p < 0.01$).

Left ventricular function after surgery. There has been considerable controversy during the past decade as to whether

coronary bypass surgery improves ventricular function. Patients with unstable angina were noted to exhibit improved left ventricular function after surgery (8), but the findings in stable angina were less clear (10,11). Earlier studies (14,15) evaluating global function at rest often failed to show improvement after surgery. The difficulty in interpreting these conflicting results is compounded because these studies did not provide information on the success of the revascularization assessed by both anatomic and perfusion methods.

Our study is the first to demonstrate that in patients with stable angina, improved regional function correlates with improved perfusion documented by both exercise myocardial scintigraphy and selective graft angiography. Rozanski et al. (9) found similar results but their assessment of regional function, utilizing radioisotope methods, was qualitative, and graft patency was not determined in their study. In our patients, every ventricular segment demonstrating improved function was perfused by a patent bypass graft and was almost always (16 of 19) associated with a normal postoperative perfusion pattern. Nearly all segments (9 of 10) that did not show improved function were associated with graft occlusion, graft stenosis or extensive disease of the native coronary artery distal to the graft insertion. Although 6 of these 10 segments that did not show improved function were interpreted to show some improvement in perfusion, it was often incomplete (5 of 6). Furthermore, analysis of the findings of graft angiography helped to clarify these otherwise potentially confusing findings (Table 2).

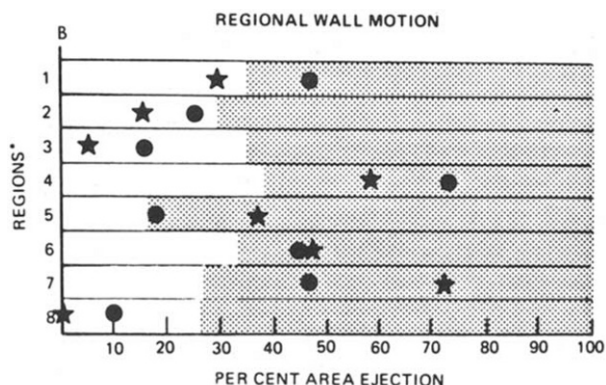
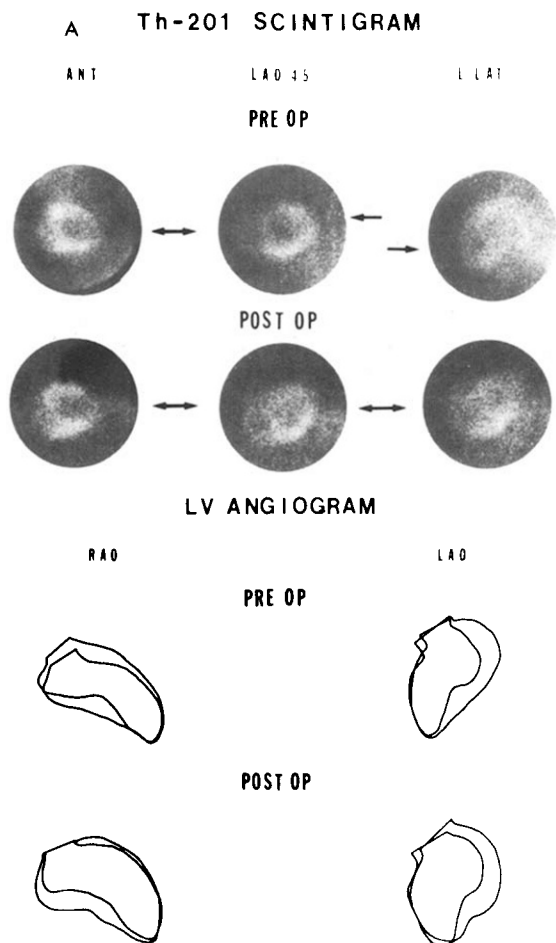


Figure 3. A, Comparison of preoperative (PRE OP) and postoperative (POST OP) thallium-201 (Th-201) scintigrams and left ventriculographic (LV) silhouettes indicates no improvement in perfusion (arrows) or wall motion. Grafts to the left anterior descending, obtuse marginal and posterior descending coronary artery were patent; however, there was severe disease in the distal left anterior descending and obtuse marginal arteries beyond the point of graft insertion. Abbreviations as before. B, Comparison of the results of preoperative (circles) and postoperative (stars) quantitative regional wall motion analysis of the ventriculographic silhouettes in A confirms no objective improvement after surgery. The stippled area represents the range of normal regional area ejection fractions.

Finally, the wall motion analysis in our study was quantitative, removing any doubt about the significance of the improved segmental function.

Stable versus unstable angina. In our study, all but two patients had stable angina pectoris before surgery. Kolibash et al. (8) found a positive correlation between improved postoperative regional function and myocardial perfusion in 14 patients with unstable angina. In their study, myocardial perfusion had to be assessed by intracoronary injection of radioactive microspheres because of the patients' clinical instability. These authors speculated that patients with stable angina would not show the same improvement. They further speculated that the controversy concerning the effect of coronary bypass surgery on left ventricular function might well be founded in the failure of earlier studies to differentiate between patients with stable and unstable angina. Our study, however, clearly shows that coronary bypass surgery can improve ventricular function in patients with stable angina as well.

Silent ischemia and stunned myocardium. Our results add to the growing body of evidence that reversible asynergy at rest is not caused by permanent myocardial scarring. Our data support the concept that these regions are composed of viable myocardium that is functionally impaired by the

effects of recent or silent ongoing ischemia (16). Our previous work (7) has shown that ventricular segments with better function after sublingual nitroglycerin or a premature ventricular contraction, and which have been shown by others (17) to contain predominantly viable myocardium, usually have a reversible perfusion deficit in the same region. The present study extends these observations in showing that successful revascularization of such segments, which improves perfusion, will permanently improve their function.

The mechanism of these functional abnormalities remains unknown. They may be the result of abnormally low perfusion at rest, which remains undetected because available methodology is insensitive. Experimentally, only minor reductions in coronary blood flow at rest are required to produce significant contractile dysfunction (18). Recently, an alternative explanation for reversible asynergy at rest has been proposed (16). In animals, short periods of intermittent but severe ischemia may "stun" the myocardium to produce protracted periods of regional dysfunction. Braunwald and Kloner (16) suggest that reversible asynergy at rest may result from repeated episodes of either symptomatic or silent ischemia. Our study does not determine if reversible asynergy at rest is the result of chronic silent ischemia or previous ischemic episodes, but it does indicate that this viable myocardium can again function normally if perfusion is improved by successful bypass surgery.

Currently, studies to determine the cause of reversible asynergy at rest are limited because clinically available methods for evaluating myocardial blood flow at rest are

too insensitive to detect the small reductions in flow that may be associated with regional dysfunction at rest. New noninvasive technologies, such as ultrafast computed tomographic scanning (19) and nuclear magnetic resonance (20), may determine whether chronic or intermittent reductions in blood flow are associated with regions of reversible resting asynergy.

Our results indicate that 40% of myocardial regions with an exercise-induced perfusion defect have a wall motion abnormality at rest. Thallium-201 myocardial perfusion scintigraphy, coupled with noninvasive assessment of regional ventricular function, appears to be a good means of identifying patients whose ventricular dysfunction can be improved by coronary bypass surgery.

Implications. We have previously reported that reversible asynergy at rest is associated with exercise-induced perfusion abnormalities. In this study, we demonstrated that normalization of these perfusion abnormalities is correlated with improvement in wall motion at rest. Our results indicate that successful revascularization by coronary bypass grafting can permanently relieve reversible ventricular asynergy, even in patients with stable angina. The results are consistent with but do not prove the hypothesis that chronic ischemia or postischemic "stunning" of the myocardium may impair regional ventricular function in the rest state. In the future, therapy for coronary artery disease might well be directed toward eliminating this ischemic manifestation as well as symptoms.

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